Antihypertensive Treatment and US Trends in Stroke Mortality, 1962 to 1980

ABSTRACT

Objectives. This study examines the association between increases in antihypertensive pharmacotherapy and declines in stroke mortality among 96 US groups stratified by race, sex, age, metropolitan status, and region from 1962 to 1980.

Methods. Data on the prevalence of controlled hypertension and socioeconomic profiles were obtained from three successive national health surveys. Stroke mortality rates were calculated using data from the National Center for Health Statistics and the Bureau of the Census. The association between controlled hypertension trends and stroke mortality declines was assessed with weighted regression.

Results. Prior to 1972, there was no association between trends in controlled hypertension and stroke mortality declines ($\beta = 0.04$, $P = .69$). After 1972, groups with larger increases in controlled hypertension experienced slower rates of decline in stroke mortality ($\beta = 0.16$, $P = .003$). Faster rates of decline were modestly but consistently related to improvements in socioeconomic indicators only for the post-1972 period.

Conclusions. These results do not support the hypothesis that increased antihypertensive pharmacotherapy has been the primary determinant of recent declines in stroke mortality. Additional studies should address the association between declining stroke mortality and trends in socioeconomic resources, dietary patterns, and cigarette smoking. (*Am J Public Health.* 1992;82:1600–1606)

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Introduction

Widespread declines in stroke mortality have been observed in the United States since the early 1900s, with variation in the magnitude and timing of declines reported by race, sex, and age group.[1–6] Although the factors that have facilitated these long-term declines have not been identified, the recent declines in stroke mortality are commonly thought to be primarily a result of increased and improved pharmacotherapy for hypertension. Given the strength of the association between elevated blood pressure and stroke,[7] together with results of randomized clinical trials that provide evidence for the efficacy of antihypertensive drugs in reducing the risk of stroke,[8,9] many health care providers, researchers, and policymakers operate under the assumption that increased and improved pharmacotherapy for hypertension is the primary determinant of the recent declines in stroke mortality. The underlying hypothesis, referred to in this study as the treatment hypothesis, is that the greater the increases in detection, treatment, and control of hypertension, the larger the decline in stroke mortality rates.

Despite clinical evidence that antihypertensive drugs reduce the risk of stroke, the impact of increased pharmacotherapy for hypertension on the declines in stroke mortality is debatable. Studies of population attributable benefit estimated that the proportion of decline in stroke mortality attributable to increased pharmacological treatment of hypertension was approximately 10% in New Zealand from 1973 to 1980, and approximately 12% in the United States from 1970 to 1980.[10,11] Another study reported no association between the rank ordering of changes in pharmacological treatment of hypertension and changes in stroke mortality from 1973 to 1980 among eight race-, sex-, and age-specific groups in the United States.[12] Our study expands upon previous studies by examining the association between temporal trends in pharmacological control of hypertension and declines in stroke mortality among 96 groups stratified by race, sex, age, metropolitan status, and region, while considering the influences of hypertension prevalence and socioeconomic profile.

Methods and Materials

Data on the prevalence and pharmacological control of hypertension, stroke mortality rates, and socioeconomic profiles were obtained for the 96 population subgroups formed by the cross tabulation of the following variables: age (45–54, 55–64, 65–74); race-sex (Black women, Black men, White women, White men); metropolitan status (metropolitan [to accommodate the different geographic units used for the survey data vs the mortality data, metropolitan status was defined as either a standard metropolitan statistical area or

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any county that included a piece of such an area], nonmetropolitan); and region (Northeast, Midwest, South, West; see Appendix A for the list of states in each region as defined by the National Center for Health Statistics). These 96 groups constitute the units of analysis for this study.

Data on the prevalence of hypertension (proportion of people who have diastolic blood pressure of at least 95 mm Hg and/or are currently using antihypertensive medication), controlled hypertension (proportion of hypertensives who reported receiving antihypertensive medication and also had controlled diastolic blood pressure levels below 95 mm Hg), and treated hypertension (proportion of hypertensives who reported receiving antihypertensive medication) were obtained from the following national health surveys: National Health and Examination Survey 1960–1962 (NHES), National Health and Nutrition Examination Survey I 1971–1974 (NHANES I), and National Health and Nutrition Examination Survey II 1976–1980 (NHANES II). In the interest of using comparable blood pressure measurements over the three surveys, hypertension was determined by the value of the first blood pressure measurement at time of examination.[13]

Each survey was a complex multistage sample survey designed to assess the health status of the civilian noninstitutionalized adult population in the United States. Participants between the ages of 45 and 74 who were both interviewed and examined are included in this study (NHES: n = 2650; NHANES I: n = 6277; NHANES II: n = 6522). The appropriate sample weights were used with SESUDAAN[14] to estimate representative proportions for each of the 96 sociodemographic groups in this study. Stein's estimator was applied to adjust for less stable estimates, which could otherwise distort the distribution, and to reduce regression to the mean of the estimated proportions across the three national health surveys.[15–17] Stein's estimator shrinks each estimated proportion toward the center of the distribution in a manner that is inversely associated with the standard deviation of the estimated proportion (see Appendix B).

For each group, the following indicators of socioeconomic profile were obtained from the national health surveys in the same manner described above: education profile (the percentage of people who received a high school education or more); income profile (the percentage of people with an annual income greater than $10 000); occupation profile (the percentage of people employed in white-collar occupations—i.e., professional, technical, and kindred workers; managerial, clerical sales, and kindred workers). Previous studies suggest that the availability of economic and social resources, as measured by census data of these indicators, is associated with patterns of cardiovascular disease mortality.[18–21]

Annual numbers of stroke deaths, based on the underlying cause of death, were obtained from the National Center for Health Statistics. Population counts were obtained from the US Bureau of the Census. Directly age-adjusted stroke mortality rates were calculated for each sociodemographic group (within 10-year age bands) by grouping the stroke deaths (*International Classification of Diseases* codes 330 to 334 for the seventh revision, codes 430 to 438 for the eighth and ninth revisions) and population counts into groups stratified by age, race, sex, metropolitan status, and region. The 1970 population was used as the standard.

The average annual absolute change in prevalence of controlled hypertension was calculated by dividing the magnitude of change from one survey to the next by the number of years that had passed from the midpoint of the first survey to the midpoint of the next survey. This method was also used to calculate change in prevalence of treated hypertension and in socioeconomic profile. The average annual percent change in stroke mortality was estimated with a piecewise log linear regression model[22] for the time periods 1962 to 1972 and 1973 to 1980.

We divided the period of observation into two (1962–1972, 1973–1980) for the following reasons: (1) stroke mortality trends among many of the sociodemographic groups in this study suggest that declines in stroke mortality accelerated roughly around 1972,[2–4] and (2) the timing of the national health examination surveys corresponds with the two time periods.

TABLE 1—Distributions of the Trends in Stroke Mortality and Controlled Hypertension among 96 Sociodemographic Groups, by Time Period

	Average Annual Percent Change in Stroke Mortality		Average Annual Absolute Change in Controlled Hypertension	
	1962–1972	1973–1980	1962–1972	1973–1980
Weighted average	–1.6	–6.5	–0.4	2.0
Median	–2.0	–6.8	–0.5	2.8
Selected percentiles				
10th	–4.3	–8.4	–1.9	–0.1
90th	–0.8	–5.1	1.1	5.3
Interdecile range	3.5	3.3	3.0	5.4

Population weighted linear regression was used to estimate the association between change in stroke mortality and change in the prevalence of controlled hypertension while adjusting for the prevalence of hypertension. The prevalence of hypertension was included in the model because it is an integral component of the exposure. The potential impact of a small increase in the proportion of hypertensives with controlled hypertension will be much larger in a group with a high prevalence of hypertension compared with a group with a low prevalence of hypertension because a larger proportion of the population will have become exposed to antihypertensive treatment. Additional analyses were performed to (1) determine the association of trends in the prevalence of treated but uncontrolled hypertension with declines in stroke mortality, (2) assess effect modification of the treatment hypothesis by category of hypertension prevalence, and (3) examine the association between socioeconomic profile and declines in stroke mortality.

Results

Larger changes in both stroke mortality and controlled hypertension occurred during the post-1972 years than during the pre-1972 years (Table 1). From 1973 to 1980, the average annual percent change in stroke mortality was –6.5% per year compared with –1.6% in the pre-1972 years. The average annual absolute change in prevalence of controlled hypertension was 2.0% during the post-1972 years compared with –0.4% in the earlier period.

The shifts in the distributions of both variables are shown in Figure 1. Each point on the scatterplot represents one of the 96 population subgroups. The values for the time period 1962 to 1972 are marked with Xs; those for 1973 to 1980 are marked with squares. Given the favorable

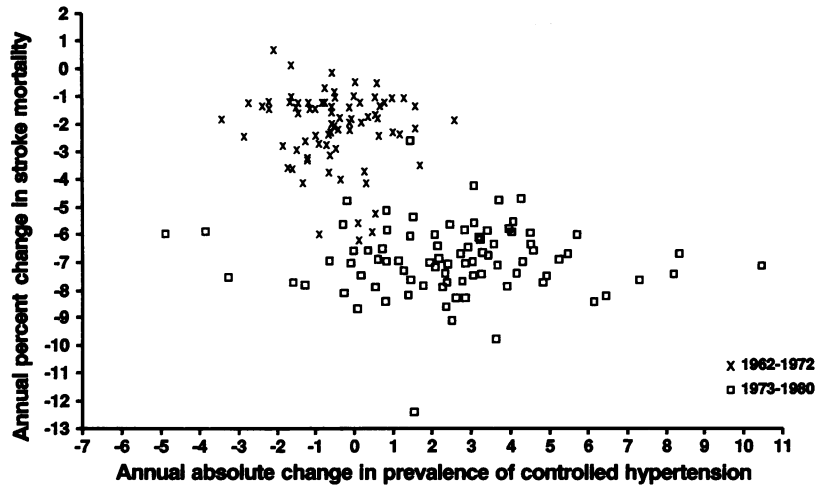

FIGURE 1—Trends in the prevalence of controlled hypertension and declines in stroke mortality among US sociodemographic groups, by time period.

TABLE 2—Association between Trends in Pharmacological Control of Hypertension and Declines in Stroke Mortality, by Time Period

	β Coefficient	SE	*P*
1962–1980			
Model 1			
Change in controlled hypertension	–0.68	.09	.0001
Prevalence of hypertension	–0.03	.02	.19
Model 2			
Change in controlled hypertension	0.13	.04	.0031
Prevalence of hypertension	–0.05	.01	.0001
Time period[a]	–5.16	.16	.0001
1962–1972			
Change in controlled hypertension	0.04	.09	.69
Prevalence of hypertension	–0.06	.01	.0001
1973–1980			
Change in controlled hypertension	0.16	.05	.003
Prevalence of hypertension	–0.05	.01	.0001

[a]Time period is an indicator variable: 0 = 1962–1972; 1 = 1973–1980.

shifts in the distributions of change in both controlled hypertension and stroke mortality, the pattern for the entire period (1962 to 1980) suggests that there was an association between trends in controlled hypertension and declines in stroke mortality. However, within each of the specified time periods, the scatterplot suggests that there was virtually no association.

Regression results quantify the observations noted in the scatterplot while adjusting for the prevalence of hypertension (Table 2). An association in favor of the treatment hypothesis is observed when the periods 1962 to 1972 and 1973 to 1980 are combined together as a single period (model 1, Table 2). The beta coefficient, –0.68, indicates that for every additional percent increase in the prevalence of controlled hypertension, the average annual rate of decline in stroke mortality increased by 0.68%. However, when time period was included in the model, it accounted for most of the previously observed association (model 2, Table 2). This implies that the overall association from 1962 to 1980 was confounded by time period (which may represent a number of factors). Therefore, the remainder of the study results are stratified by time period.

During the pre-1972 period, there was virtually no association between the trends in controlled hypertension and the declines in stroke mortality among the 96 sociodemographic groups (β = 0.04, P = .69; Table 2). During the post-1972 years, the groups with the largest increases in the prevalence of controlled hypertension experienced slightly slower rates of decline in stroke mortality (β = 0.16, P = .003). This beta coefficient for the later period indicates that, for every additional percent increase in the prevalence of controlled hypertension, the average annual rate of decline in stroke mortality slowed by 0.16%. During both the pre- and post-1972 years, the groups with the higher prevalence of hypertension at the beginning of the period experienced the larger declines in stroke mortality (β = –0.06 and –0.05 for each time period, respectively) (Table 2).

The magnitude of acceleration in stroke declines from the pre-1972 period to the post-1972 period (measured as the difference between the rate of decline in the two periods) was regressed upon the change in controlled hypertension from NHES to NHANES II. The results indicate that groups with the larger acceleration in stroke mortality declines did not show larger changes in controlled hypertension (β = 0.03, P = .04).

Examination of the association between trends in prevalence of treated hypertension (regardless of blood pressure control) and declines in stroke mortality (Table 3) produced results very similar to those presented for trends in controlled hypertension. Because control of hypertension should have a larger impact on the risk of stroke than should treatment per se, the remainder of this study focuses on the association between trends in controlled hypertension and declines in stroke mortality.

A suggestion of effect modification by prevalence of hypertension is observed in Table 4. Within each time period, the beta coefficient in favor of the treatment hypothesis occurs only among the groups in the highest tertile.

The association between trends in socioeconomic profile and declines in stroke mortality varied by time period (Table 5). In the first period (1962 to 1972), groups with the largest increases in education and income profiles experienced the slowest rates of decline in stroke mortality (i.e., a 1% increase in the proportion of high school graduates and families with annual incomes above $10 000 was associated with a slowing in the average annual rate of decline in stroke mortality of 0.30% and 0.24%, respectively). In the second period (1973 to 1980), the pattern was reversed. Groups with the largest increases in all three components of socioeconomic profile experienced modestly yet consistently faster rates of decline in stroke mortality

(β = –0.11, –0.03, and –0.04 for education, income, and occupation profiles, respectively).

Discussion

The widespread declines in stroke mortality among many sociodemographic groups in the United States suggest that equally widespread changes have occurred in population-level conditions that influence the occurrence of stroke deaths. Investigation of the association between changes in population-level conditions and declines in stroke mortality requires an aggregate-level study with a group—rather than an individual—as the unit of analysis. Although aggregate-level studies are popularly used to make inferences about associations at the individual level,[23,24] the results of this study pertain specifically to population-level associations and are not meant to be interpreted as proxies for individual-level associations.[25,26]

The results of this study contribute to a growing body of literature that suggests that, although pharmacological treatment of hypertension may have contributed to the declines in stroke mortality, it may not have been the primary determinant between 1962 and 1980.[10–12] Within the two time periods examined (1962 to 1972 and 1973 to 1980), groups with the largest increases in treatment and control of hypertension did not experience the largest declines in stroke mortality. In the pre-1972 period, there was virtually no association between increases in the prevalence of controlled hypertension and declines in stroke mortality among the 96 sociodemographic groups, while in the post-1972 period, the data suggest that groups with larger increases in controlled hypertension had slightly slower declines in stroke mortality.

When the data are stratified by prevalence of hypertension, there is some evidence in favor of the treatment hypothesis among groups with the highest prevalence of hypertension. Relationships in groups with lower prevalence are not consistent with the treatment hypothesis. This is noteworthy because groups with higher prevalences of hypertension are more likely to benefit from antihypertensive treatment programs.

The discrepancy in the results obtained when examining the association over the entire period as opposed to the association within each time period can be attributed to the major shifts in the distributions of trends in both stroke mortality and controlled hypertension. The most likely candidate for explaining the shift in controlled hypertension is the establishment in 1972 of the National High Blood Pressure Education Program, a government-sponsored coalition of private and public agencies working to increase the detection, treatment, and control of hypertension in the United States. There is little doubt that the efforts of this program have contributed to improved pharmacological management of hypertension. However, if increased pharmacological control of hypertension is to be considered the primary determinant of declines in stroke mortality, as much of the literature claims, the lack of association between trends in controlled hypertension and declines in stroke mortality within each time period remains to be

TABLE 3—Association between Trends in Pharmacological Treatment of Hypertension and Declines in Stroke Mortality, by Time Period

	β Coefficient	SE	*P*
1962–1980			
Model 1			
Change in treated hypertension	–0.73	.08	.0001
Prevalence of hypertension	–0.04	.02	.19
Model 2			
Change in treated hypertension	–0.0002	.04	.96
Prevalence of hypertension	–0.05	.01	.0001
Time period[a]	–4.80	.17	.0001
1962–1972			
Change in treated hypertension	0.03	.08	.74
Prevalence of hypertension	–0.05	.01	.0001
1973–1980			
Change in treated hypertension	0.02	.05	.74
Prevalence of hypertension	–0.04	.01	.0001

[a]Time period is an indicator variable: 0 = 1962–1972; 1 = 1973–1980.

TABLE 4—Association between Trends in Pharmacological Control of Hypertension and Declines in Stroke Mortality,[a] Stratified by Category of Hypertension Prevalence for Each Time Period

	β Coefficient	SE	*P*
1962–1972, category of hypertension prevalence[b]			
≤23.4%	0.07	.14	.62
23.5%–31.6%	0.05	.12	.67
≥31.7%	–0.09	.25	.73
1973–1980, category of hypertension prevalence[c]			
≤34.0%	0.21	.09	.02
34.1%–45.0%	0.16	.10	.10
≥45.1%	–0.08	.08	.34

[a] Baseline prevalence of hypertension is included in the model.
[b] Categories are tertiles of the hypertension prevalence distribution in NHES.
[c] Categories are tertiles of the hypertension prevalence distribution in NHANES I.

TABLE 5—Association between Trends in Socioeconomic Profile and Declines in Stroke Mortality, for Each Time Period

	1962–1972		1973–1980	
Component of Socioeconomic Profile	β Coefficient	SE	β Coefficient	SE
Change in education profile	0.30*	.15	–0.11	.09
Change in income profile	0.24**	.06	–0.03	.07
Change in occupation profile	–0.12	.08	–0.04	.05

*$.05 < P \leq .10$; **$P \leq .01$.

explained, as does the lack of association between accelerated stroke mortality declines and increased controlled hypertension.

The smaller amount of variability in the distributions of changing prevalence of controlled hypertension and stroke mortality within each period vs the combined periods could make it more difficult to detect an association within each period. In addition, the instability of proportions and rates due to small numbers among some of the 96 sociodemographic groups could restrict the ability to detect an association. Other potential sources of error in this study include nonresponse bias for the variables obtained from the national health surveys[27] and variation in the accuracy of stroke mortality diagnosis due to use of death certificate data.[28–30] Although these factors are not likely to occur randomly, there is not enough information to speculate upon the direction of potential bias.

The lack of available data on the severity of hypertension at the onset of pharmacological treatment may also hamper the ability to evaluate fully the treatment hypothesis. The magnitude of reduction in absolute risk is greater among severe hypertensives than among mild and moderate hypertensives.[31] Therefore, according to the treatment hypothesis, groups with larger proportions of severe hypertensives at the onset of treatment may be expected to have larger declines in stroke mortality. Alternatively, groups with larger prevalences of severe hypertension among the nontreated hypertensives may have slower stroke mortality declines even if they have large proportions of hypertensives with controlled hypertension.

Inclusion of data on trends in cigarette smoking would also improve the evaluation of the treatment hypothesis and our understanding of the trends in stroke mortality.[32–34] Although, during the earlier period of observation in this study, the prevalence of smoking among some sociodemographic groups was increasing while stroke mortality rates were decreasing, the recent declines in smoking among several sociodemographic groups[35–38] may have contributed to the declines in stroke mortality for those groups. Unfortunately, comparable data on smoking trends were not available for the 96 sociodemographic groups in this study.

Alternative explanations for the lack of evidence in favor of the treatment hypothesis deal with the historical context within which the antihypertensive programs were introduced and the differential outreach of such programs among the sociodemographic groups included in this study. Declines in stroke mortality among both Blacks and Whites were occurring decades prior to the introduction of antihypertensive treatment.[1–4] Therefore, factors that contributed to the long-term declines in stroke mortality may have had a stronger impact on the recent trends in stroke mortality than did the recent increases in pharmacological treatment of hypertension. This seems especially plausible given patterns of change in controlled hypertension and stroke mortality among sociodemographic groups. For instance, from 1973 to 1980, White men and Black women experienced the fastest average annual rate of decline in stroke mortality (7.7% for Black women and 7.0% for White men, compared with 6.1% and 6.3% for White women and Black men, respectively). White men achieved these large declines in stroke mortality with an average annual increase of only 1.1% in the prevalence of controlled hypertension, whereas the increase for Black women was 4.5%—four times larger than that of White men. Consequently, other factors may operate at the population level that either add to or detract from the effectiveness of increased antihypertensive pharmacotherapy on declines in stroke mortality or that influence the rates of stroke mortality directly.

Modest associations of the temporal trends in socioeconomic profile with the declining stroke mortality rates were observed from 1973 to 1980. Larger associations were observed (but not presented) between the level of stroke mortality and indicators of socioeconomic profile, as has been reported in other studies.[21,39–41] These results lend further support to the view that socioeconomic conditions define the context within which the distributions of physiological and behavioral risk factors are determined.[42–44] Further studies are needed to examine the distributions of identified risk factors for essential hypertension[45,46] (i.e., diet, physical activity, psychosocial stressors) by socioeconomic indicators, and to better elucidate the mechanisms that link environmental stimuli to physiological responses associated with hypertension and with stroke directly.

The implications of the results from this study neither address nor challenge the efficacy of antihypertensive drugs to reduce the risk of stroke among individuals under conditions of clinical and community trials. Instead, the results are pertinent to an understanding of the impact of the high-risk intervention strategy on the declining stroke mortality rates from a public health perspective. It appears that, among the sociodemographic groups in this study, the mass treatment approach is not the principal determinant of the declines in stroke mortality. This does not imply that clinicians should not treat hypertensives with antihypertensive agents, but, from a public health perspective, it does support the idea that we cannot rely on mass treatment of hypertension to bring about the greatest declines in stroke mortality. Further support for this conclusion comes from the fact that the largest population attributable risk for all cause mortality occurs in the high normal to mild hypertension range of the blood pressure distribution.[47] While this group contributes the most to excess mortality rates, it benefits the least from the high-risk/mass treatment approach.

Although the notion was once considered a heresy, it is now widely accepted that environmental improvements in sanitation, nutrition, and housing had a larger impact on past declines in infectious disease mortality than did specific medical measures.[48] The present results, as well as evidence of long-term declines in stroke mortality prior to the development and widespread dissemination of antihypertensive drugs, suggest a more contemporary version of this old debate about the determinants of changes in mass disease. It is, of course, in keeping with basic public health tenets that environmental changes affecting entire populations are likely to have a greater impact than interventions that target individuals one at a time and depend on access and compliance for their success.[49]

From a public health perspective, further attention should be given to understanding the role of secular trends in other characteristics—e.g., educational resources, dietary patterns, occupational opportunities, cigarette smoking, and living conditions—that may have influenced the declines in stroke mortality. A better understanding of the combinations of conditions that have facilitated the declines in stroke mortality is important for planning the most effective intervention strategies to further reduce the levels of stroke mortality among all sociodemographic groups in the United States. □

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(See appendixes on next page.)

APPENDIX A—The Four Geographic Regions of the United States, as Defined by the National Center for Health Statistics

Northeast:	Pennsylvania, New Jersey, Connecticut, Rhode Island, Massachusetts, New York, Vermont, New Hampshire, Maine
Midwest:	Ohio, Michigan, Indiana, Illinois, Wisconsin, Minnesota, Iowa, Missouri
South:	Delaware, Maryland, Virginia, West Virginia, Kentucky, Arkansas, Tennessee, North Carolina, South Carolina, Georgia, Florida, Alabama, Mississippi, Louisiana, District of Columbia
West:	Washington, Oregon, Idaho, Montana, Wyoming, Colorado, Utah, Nevada, California, Arizona, New Mexico, Texas, Oklahoma, Kansas, Nebraska, South Dakota, North Dakota

APPENDIX B—The Equation for the Application of Stein's Estimator

$$p^*_i = (1 - c_i)\,\hat{p}_i + (c_i)\bar{p}_t,$$

where $_i$ = sociodemographic group (a cell),
p^*_i = weighted cell-specific proportion,
$\hat{p}_i$ = cell-specific estimated proportion,
$\bar{p}_t$ = average estimated proportion for all 96 groups combined, and
c_i = the empirical Bayes shrinkage factor.

The empirical Bayes shrinkage factor is the weight as described in the text and will always be a number between 0 and 1. The derivation of c is explained in detail in an article by Davis et al.[17] For this study, SAS program was written to calculate c for each survey variable in each of the three national health surveys.